Iranian Heart Journal; 2021; 22 (1)

Original Article

Echocardiographic Screening for Myocardial Dysfunction Among Asphyxiated Newborns

Krishnananda Nayak[1], PhD; Sara Varghese[2], MS; Abdul Razak[3*], MD; Megha A[1], MS; Tom Devasia[3], MD; Leslie Edward Lewis[4], MD; Sridevi Prabhu[1], MS

ABSTRACT

Background: Perinatal asphyxia/ischemia is an interruption in the availability of oxygen supply to the myocardium. We sought to assess myocardial function among asphyxiated and non-asphyxiated neonates using echocardiography.

Methods: The present case-control study was undertaken at the neonatal intensive care unit (NICU) of a tertiary care hospital. Neonates with asphyxia who were admitted to the NICU were included in the study. The diagnosis of asphyxia was established on the basis of the APGAR score at 1 and 5 minutes. Myocardial function was assessed in terms of serum cardiac troponin I (CTnI) and different echocardiographic parameters such as M-Mode, Doppler flow parameters, and tissue Doppler imaging.

Results: Thirty asphyxiated (the case group) and 30 non-asphyxiated (the control group) term neonates were enrolled in the study. The asphyxia group had significantly lower ($P < 0.001$) 1- and 5-minute APGAR scores than the controls. The asphyxia group had a higher serum CTnI value. The differences between the 2 groups concerning M-Mode parameters in the first 12 hours of life were statistically significant ($P < 0.001$). The case and control groups were also statistically significantly different regarding left atrial pressure at 48 hours following birth, calculated as a ratio of E/e' ($P < 0.001$). The differences between the 2 study groups at 24 hours after birth as regards tissue Doppler parameters, including left ventricular ejection time and left ventricular Tei index, were also statistically significant ($P < 0.001$). The case and control groups also showed statistically meaningful differences concerning right ventricular ejection period/ ejection time at 24 hours after birth ($P < 0.002$).

Conclusions: It can be concluded that the myocardial function assessment using CTnI and different echocardiographic techniques is the most sensitive and specific method in the detection of ischemic cardiac injury in asphyxiated newborns. ***(Iranian Heart Journal 2021; 22(1): 91-99)***

KEYWORDS: Asphyxia, APGAR score, Newborn, Troponin

[1] Department of Cardiovascular Technology, Manipal College of Health Professions, Manipal Academy of Higher Education, Manipal, India.
[2] Department of Cardiovascular Technology, School of Allied Health Science, Manipal Academy of Higher Education, Manipal, India.
[3] Department of Cardiology, Kasturba Medical College, Manipal Academy of Higher Education, Manipal, India.
[4] Department of Pediatrics, Kasturba Medical College, Manipal Academy of Higher Education, Manipal, India.

***Corresponding Author:** Abdul Razak, MD; Department of Cardiology, Kasturba Medical College, Manipal Academy of Higher Education, Manipal, India.
Email: razakkaup@gmail.com **Tel:** +919448252641

Received: September 19, 2020 **Accepted:** October 28, 2020

Iranian Heart Journal; 2021; 22 (1)

Perinatal asphyxia is a temporary interruption in the availability of oxygen, resulting in inadequate tissue perfusion. Perinatal asphyxia is also termed "hypoxic/ischemic encephalopathy". In neonates born at term, perinatal asphyxia occurs in 2 to 10 per 1000, and this ratio is comparatively high in neonates who are born prematurely.[1] Asphyxia occurs when there is an interruption in blood-gas exchange, resulting in a lack of oxygen (hypoxemia) and the accumulation of carbon dioxide (hypercapnia). The reduction in oxygen and blood supply (ischemic hypoxia) shows a cascade of biochemical changes in the body, which leads to neuronal cell death and brain damage. In neonatal post-asphyxia syndrome, multiple organ dysfunction is the most common feature. Perinatal asphyxia is considered to be an important cause for short-and long-term morbidity and mortality in neonates.[2, 3] Perinatal birth asphyxia may have maternal or fetal causes. Those who survive asphyxia at birth may have a probability of developing central nervous system complications, including permanent neurological handicaps, cerebral palsy, visual defects, mental retardation, cognitive impairment, and epilepsy.[3, 4] Myocardial injury in perinatal asphyxiated neonates with hypoxic/ischemic encephalopathy can be appropriately diagnosed through significant clinical findings. Several early markers have been assessed to identify asphyxiated newborns; they include cardiac troponin I (CTnI), electrocardiography, tissue Doppler echocardiography, computed tomography, and tissue Doppler imaging (TDI).[5] The level of CTnI can be used as a reliable early predictor of poor outcomes in neonates with perinatal asphyxia.[6] Echocardiography is used as a diagnostic tool in asphyxiated neonates to rule out any congenital heart diseases, to assess associated primary pulmonary artery hypertension, and to assess the qualitative and quantitative degree of myocardial dysfunction.[7] Several other clinical methods are also drawn upon for diagnosis and prognosis in perinatal asphyxia; they include non-reassuring fetal heart rates, which indicate fetal distress and a prolonged labor time; meconium-stained amniotic fluid; low APGAR score levels; and academia.

Accordingly, we designed the present study to investigate myocardial dysfunction among asphyxiated and non-asphyxiated neonates using echocardiography.

METHODS

The current case-control investigation was performed at the neonatal intensive care unit (NICU) of a tertiary care hospital. Postnatal asphyxiated babies within 12 hours were considered the case group, while age- and gender-matched healthy neonates with normal cardiac findings selected via the frequency matching method were recruited as controls. Infants with metabolic disorders, chromosomal abnormalities, *in utero* infections, severe diseases, and complex congenital heart diseases were excluded from the study. The study protocol was approved by the institutional ethics committee, and written informed consent was obtained from all the children's parents before the inclusion of their child in the study.

Echocardiographic screening was done within the first 12 hours of birth, followed by 24 and 48 hours after birth. The diagnosis of asphyxia was established based on the APGAR score at 1 and 5 minutes. Different cardiac views were stored as short clips for subsequent off-line analysis. Serum concentrations of CTnI were measured in both case and control groups within the first 12 hours of life via the ECLIA method according to the manufacturer's cutoff point

(<0.002 ng/mL). The neonates underwent transthoracic echocardiography with the PHILIPS CX50 with S12-8MHz transducers. Myocardial involvement was recorded in both groups by using different echocardiographic parameters, including 2D, M-Mode, Doppler flow, and TDI. M-Mode parameters, consisting of left ventricular end-diastolic diameter (LVEDD), left ventricular end-systolic diameter (LVESD), ejection fraction (EF), fractional shortening (FS), LV septum thickness, and maximum aortic cusp separation (MACS), were assessed. Using TDI, other parameters, namely LV pre-ejection period, LV ejection time, LV acceleration time, right ventricular (RV) pre-ejection period, RV ejection time, and RV acceleration time, were calculated.

Statistical Analysis

The statistical analyses were carried out using a Microsoft Excel Spreadsheet, version 2007, (Microsoft Corp, Seattle, Washington). Values were expressed as mean±SD or percentages. The analyses were performed using the independent sample *t*-test with SPSS software, version 20. A *P* value of less than 0.05 was considered statistically significant. Echocardiographic parameters at different time points were compared between the case and control groups using mixed analysis of variance (ANOVA) ensuring that the continuous variables were normally distributed.

RESULTS

Sixty neonates were enrolled in the current study with an equal sex ratio. The difference in terms of the mean gestational age was statistically significant between the asphyxia group and the control group (32.8 ± 4.9 vs 37.4 ± 1.5 wk; $P < 0.001$). Birth weight in the asphyxia group was statistically significantly less than that in the control group (2328.73 ± 616.275 vs 2807.03 ± 257.039 g; $P < 0.001$). The asphyxia group had significantly lower 1- and 5-minute APGAR scores than the controls ($P < 0.001$). The mean serum CTnI level was significantly higher in the asphyxia group (> 0.002 ng/mL). Table 1 shows comparisons between the 2 study groups concerning demographic and clinical parameters.

Table 2 presents conventional echocardiographic measurements. The M-Mode parameters, comprised of LVEDD, LVESD, EF, FS, LV septum thickness, and MACS, were higher in the asphyxia group than in the control group. Further, there were statistically significant differences between the 2 groups in the first 12 hours of life regarding the M-Mode parameters of LVEDD, LVESD, and FS ($P < 0.001$). LVEF was significantly lower in the asphyxia group in the first 12 hours ($P < 0.001$) and 24 hours ($P < 0.004$). Statistically meaningful differences were also detected in terms of LV septum thickness and MACS between the case and control groups ($P < 0.001$). The differences between the asphyxiated and non-asphyxiated groups vis-à-vis aortic diameter, left atrial dimension, MACS, LVEDD, LVESD, EF, FS, LV septum thickness, and LV posterior wall thickness were also statistically significant ($P < 0.001$).

RV diastolic dimension showed a statistically significant difference between the 2 groups at 24 hours ($P < 0.002$) and 48 hours ($P < 0.001$). Moreover, RV systolic dimension was statistically significantly different between the case and control groups at 48 hours ($P < 0.002$). Also statistically meaningfully different between the asphyxiated and non-asphyxiated groups was RV free wall thickness ($P < 0.001$). Additionally, there was a significant difference with regard to RV hypertrophy between the asphyxia group and the control group ($P < 0.001$). Tricuspid annular plane systolic excursion (TAPSE), which is an M-

Mode parameter that indicates RV function, was statistically significantly different between the groups ($P < 0.001$). Furthermore, the differences between the case and control groups showed statistical significance as regards RV systolic pressure

The results also revealed differences of statistical significance between the 2 groups of neonates in regard to RV diastolic dimension, RV free wall thickness, TAPSE, and RV systolic pressure ($P < 0.001$).

Table 1: Demographic and clinical parameters of the 2 study groups

Parameters	Case	Control	*P* value
Gestational Age ($^{+}$wk)	32.8 ±4.9	37.4 ± 1.5	<0.001
Birth Weight (g)	2328.73 ± 616.275	2807.03 ± 257.039	<0.001
APGAR Score at 1 min	3.6 ± 1.19	8.37 ± 0.66	<0.001
APGAR Score at 5 mine	5.5 ± 1.04	9.37 ± 0.615	<0.001
Serum Cardiac Troponin T (ng/mL)	0.28 ± 0.2	-	<0.001

Table 2: Conventional echocardiographic measurements

	Case			Control			*P* value			Pa
	12 h	24 h	48 h	12 h	24 h	48 h	12 h	24 h	48 h	
LV Parameters										
Aortic diameter	8.4±1.2	8.5±1.5	8.4±1.1	7.9±0.1	7.9±0.8	7.9±0.9	0.124	0.44	0.074	0.001
LA dimension (mm)	10.4±1.5	10.4±1.5	10.5±1.9	9.4±1.2	9.5±1.2	9.6±0.9	0.006	0.018	0.027	0.01
MACS (mm)	5.5±0.9	5.5±0.7	5.7±0.9	4.6±0.6	4.7±0.5	4.6±0.4	<0.001	< 0.001	< 0.001	0.001
LVEDD (mm)	15.13 ± 2.09	15.1 ± 2.2	14.7 ± 2.2	13.1 ± 1.6	13.8 ± 1.6	13.4 ± 1.5	<0.001	0.013	0.008	0.001
LVESD (mm)	9.7±2.2	9.4±2.3	9.1±2.0	8.1±1.4	8.4±1.5	8.3±1.4	0.001	0.052	0.084	0.001
EF (%)	62.8 ±7.7	64.3 ± 7.1	66.6 ± 3.9	68.9 ± 4.2	68.6 ± 3.2	68.1 ± 3.0	<0.001	0.004	0.134	0.01
FS (%)	33.7 ± 6.03	34.8 ± 5.2	36.4 ± 3.2	38.7 ± 3.8	38.8 ± 3.5	38.2 ± 2.7	< 0.001	0.037	0.132	0.001
CO (%)	0.13 ± 0.34	0.26 ± 0.44	0.12 ± 0.32	0.1 ± 0.3	0.07 ± 0.25	0.07 ± 0.25	0.694	0.047	0.532	0.001
LV septum thickness (mm)	4.2±0.6	4.3± 0.8	4.4± 0.6	3.4±0.5	3.4± 0.5	3.5± 0.5	<0.001	<0.001	<0.001	0.001
LV posterior wall thickness (mm)	3.4±0.6	3.4 ±1.0	3.3±0.8	3 ± 0.37	3 ± 0.37	2.9±0.2	0.006	0.034	0.017	0.002
RV Parameters										
MPA diameter mm)	7.9± 1.7	8 ±1.3	8.2±1.7	7.1±0.9	7.1±0.95	7.2±0.9	0.013	0.007	0.006	0.738
RVDd (mm)	11.8±2.4	12.4±2.2	12.6±2.2	11.07±1.5	10.9±1.2	10.8±1.1	0.162	0.002	<0.001	0.002
RVSd (mm)	9.7 ± 2.3	14±19.7	10.1±2.0	9.07±1.5	8.8 ± 1.4	8.6±1.2	0.223	0.157	0.002	0.06
RV free wall thickness mm)	4.4±1.2	4.2±0.9	4.2±0.8	3.0±0.2	2.9±0.2	2.9±0.2	<0.001	<0.001	<0.001	0.001
TAPSE (mm)	6.8±1.3	6.7±1.6	6.7± 1	7.8± 1.1	8.03±1.1	8.2±1.1	0.002	0.001	<0.001	0.001
RVSP	40.9± 27.6	35.9± 16.4	32.1± 13.9	16.6 ± 6.3	17.8 ± 6.4	15.6 ± 5.7	<0.001	<0.001	<0.001	0.001

LV, Left ventricle; LA, Left atrium; MACS, Maximum aortic cusp separation; LVEDD, Left ventricular end-diastolic diameter; LVESD, Left ventricular end-systolic diameter; EF, Ejection fraction; FS, Fractional shortening; CO, Cardiac output; RV, Right ventricle; MPA, Main pulmonary Artery; RVDd, Right ventricular diastolic dimension; RVSd, Right ventricular systolic Dimension; TAPSE, Tricuspid annular plane systolic excursion; RVSP, Right ventricular systolic pressure

Left atrial pressure, calculated as a ratio of E/e', was statistically significantly different between the 2 groups at 48 hours ($P < 0.001$). Also statistically meaningfully different between the groups were mitral annular velocity and left atrial pressure ($P < 0.001$) (Table 3).

Tissue Doppler parameters, including LV ejection time, showed a statistically significant difference between the case and control groups at 24 hours after birth ($P < 0.001$) (Table 4). Moreover, LV Tei index showed a statistically significant difference between the asphyxiated and non-asphyxiated neonates at 24 hours following birth ($P < 0.001$). The groups showed a statistically significant difference as regards RV ejection period/ejection time at 24 hours after birth ($P < 0.002$). The difference between the 2 groups of neonates with respect to RV early diastolic wave was also statistically significant at 24 and 48 hours of age ($P < 0.001$).

A noteworthy case was that of a neonate with an APGAR score of 3/10 at 1 minute and 4/10 at 5 minutes of life. The neonate was confirmed with birth asphyxia (hypoxic/ischemic encephalopathy: Stage II). The CTnI level was greater than 0.020 ng/mL. Fetal perinatal asphyxia persisted. The neonate underwent routine echocardiography on the first day of life; the results showed good LV systolic function, a small patent foramen ovale, and moderate mitral regurgitation. The moderate mitral regurgitation persisted on the first day of life without any significant cause, but the mitral valve and LV function appeared to be completely normal (Fig. 1). Follow-up echocardiography was done on the second day of the neonate's life, and the results demonstrated a reduction in the mitral regurgitation compared with the first day (Fig. 2). The moderate mitral regurgitation decreased to trivial mitral regurgitation without any cause.

Table 3: Doppler parameters (LV and RV)

Doppler Parameters	Case			Control			*P* value			P[a]
	12 h	24 h	48 h	12 h	24 h	48 h	12 h	24 h	48 h	
Mitral E (m/s)	0.43±0.1	0.47±0.1	0.44±0.1	0.4±0.1	0.45±0.1	0.43±0.1	0.321	0.589	0.771	0.37
Mitral A (m/s)	0.6±0.09	0.49±0.13	0.5 ± 0.1	0.5±0.1	0.48±0.13	0.5± 0.1	0.017	0.957	0.920	0.20
E /A Ratio	0.9 ± 0.4	1.1 ± 0.4	0.89±0.2	0.8±0.2	0.98±0.3	0.89±0.3	0.125	0.389	0.979	Ns
Tricuspid E(m/s)	-	-	-	-	0.4 ± 0.1	0.44±0.1	0.947	0.260	0.260	0.28
LAP (mm Hg)	7.8±3.2	9.6±2.4	8.9±2.3	7.3±2.02	8.5±2.8	7.0±1.3	0.133	0.124	<0.001	0.01

LV, Left ventricle; RV, Right ventricle; E wave, Peak early diastolic inflow velocity; A wave, Peak late diastolic inflow velocity; LAP, Left atrial pressure

Table 4: Tissue Doppler (LV and RV parameters)

	Case			Control			*P* value			P[a]
	12 h	24 h	48 h	12 h	24 h	48 h	12 h	24 h	48 h	
LV Parameters										
LV IVRT	0.1±0.12	0.09±0.1	0.08±0.1	0.06±0.01	0.06±0.01	0.06±0.01	0.178	0.268	0.314	0.002
LV IVCT	0.07± 0.06	0.08± 0.08	0.08± 0.08	0.06± 0.01	0.06± 0.01	0.06± 0.01	0.240	0.193	0.241	0.69
LV ET	0.18 ± 0.03	0.19 ± 0.02	0.17 ± 0.03	0.16 ± 0.02	0.16 ± 0.02	0.16 ± 0.02	0.022	<0.001	0.344	0.01
LV Tei Index	0.6±0.16	0.61±0.1	0.67±0.1	0.99±1.5	0.77±0.1	0.75±0.1	0.204	0.001	0.106	0.03
LV EP/ET	0.34 ± 0.07	0.34 ± 0.08	0.35 ± 0.09	0.34 ± 0.07	0.36 ± 0.08	0.37 ± 0.07	0.639	0.303	0.486	0.007

LV AT/ET	0.35 ± 0.08	0.32 ± 0.05	0.33 ± 0.06	0.36 ± 0.06	0.35 ± 0.06	0.37 ±0.079	0.723	0.057	0.039	0.05
IVS Em	0.04 ± 0.01	0.05 ± 0.07	0.26 ± 1.1	0.06 ± 0.03	0.06 ±0.029	0.06 ± 0.02	0.087	0.685	0.342	0.001
IVS Am	0.06 ± 0.1	0.05 ± 0.01	0.05 ± 0.01	0.05 ± 0.02	0.05 ± 0.02	0.05 ± 0.02	0.619	0.623	0.398	0.001
IVS S	0.04 ± 0.01	0.04 ± 0.01	0.04 ± 0.01	0.04 ±0.009	0.04 ± 0.02	0.05 ± 0.06	0.619	0.178	0.222	0.01
LV Em	0.05 ±0 .01	0.05 ± 0.01	0.05 ± 0.01	0.11 ± 0.18	0.08 ± 0.12	0.09 ± 0.13	0.0909	0.265	0.209	0.15
LV Am	0.05 ± 0.018	0.06 ± 0.01	0.06 ± 0.02	0.06 ±0.019	0.06 ± 0.01	0.06 ± 0.02	0.837	1.0	0.641	ns
LV S	0.05 ± 0.013	0.04 ± 0.01	0.05 ± 0.01	0.05 ±0.012	0.05 ± 0.01	0.05 ± 0.01	0.603	0.377	0.945	0.88
RV Parameters										
RV EP/ET	0.31 ± 0.08	0.33 ± 0.08	0.35 ± 0.09	0.37 ± 0.07	0.4 ± 0.06	0.39 ± 0.08	0.008	0.002	0.173	0.002
RV AT/ET	0.37 ± 0.09	0.37 ± 0.09	0.39 ± 0.1	0.44 ± 0.11	0.44 ± 0.08	0.44 ± 0.09	0.008	0.011	0.035	0.0013
RV Tei Index	0.63 ± 0.34	0.65 ± 0.26	0.75 ± 0.35	0.71 ± 0.18	0.79 ± 0.57	0.75 ± 0.16	0.281	0.260	0.989	0.001
RV Em	0.06 ± 0.02	0.06 ± 0.01	0.05 ± 0.01	0.08 ± 0.04	0.09 ± 0.04	0.08 ± 0.03	0.010	0.001	<0.001	0.0004
RV Am	0.11 ± 0.17	0.09 ± 0.03	0.08 ± 0.02	0.09 ±0.035	0.09 ± 0.03	0.08 ± 0.03	0.517	0.724	0.249	0.001
RV S	0.39 ± 1.8	0.34 ± 1.4	0.36 ± 1.4	0.06 ± 0.01	0.07 ± 0.02	0.09 ± 0.13	0.320	0.294	0.307	0.29

LV, Left ventricle; IVRT, Isovolumetric relaxation time; IVCT, Isovolumetric contraction time; ET, Ejection time; EP, Ejection period; AT, Acceleration time; IVS, Interventricular septum; Em, Early diastolic wave; Am, Late diastolic wave; S, Systolic wave; RV, Right ventricle; EP, Ejection period; ET, Ejection time; AT, Acceleration time; ns, not significant

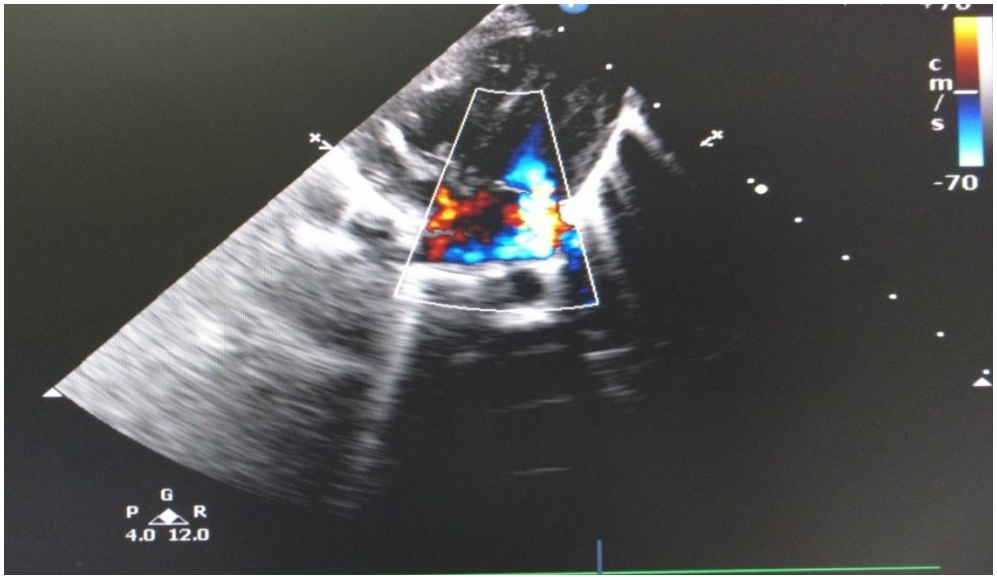

Figure 1: Color Doppler in the apical 4-chamber view shows moderate mitral regurgitation (first day of life).

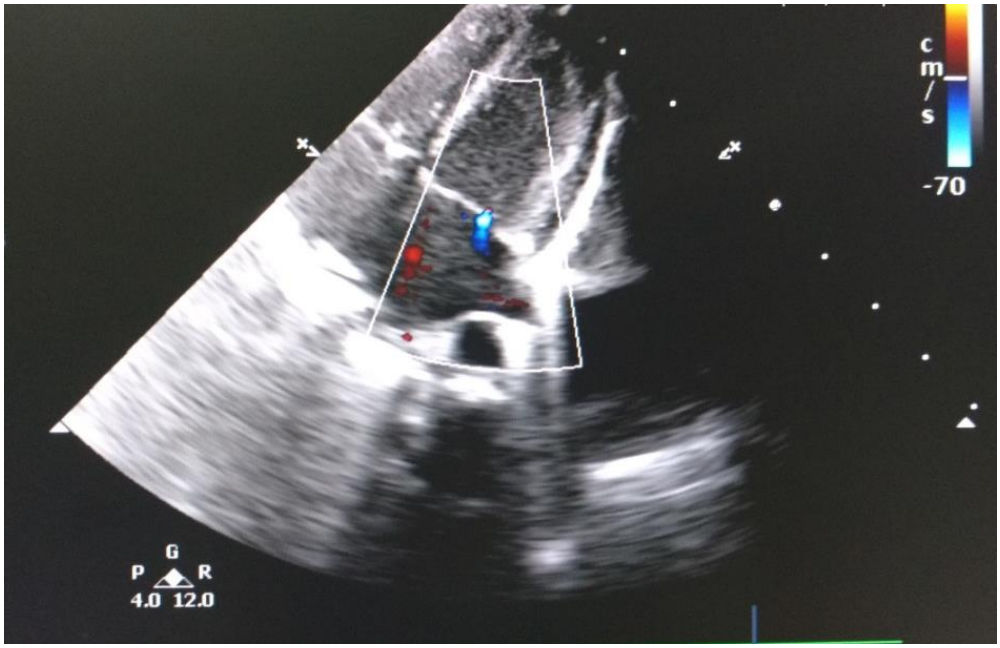

Figure 2: Color doppler in the apical 4-chamber view shows trivial mitral regurgitation (second day of life).

DISCUSSION

The detection of myocardial abnormalities is complicated in neonates because the clinical utility of clinical signs, echocardiographic measurements, and cardiac enzymes remains unknown. There are some studies on the effects of perinatal asphyxia and the diagnostic value of these tests in the determination of myocardium function in neonates.[8, 9] Hence, we performed the present study to evaluate myocardial dysfunction in terms of serum CTnI and different echocardiographic parameters among asphyxiated and non-asphyxiated neonates.

According to our results, the differences between the 2 study groups of asphyxiated and non-asphyxiated neonates regarding gestational age and birth weight were statistically significant ($P < 0.001$). We drew upon the Tei index as a parameter for the assessment of overall global and regional myocardial performance in both groups and also assessed the relationship between these measurements and the serum CTnI concentration, which is a highly specific marker in myocardial injury.[10]

The asphyxia group had lower 1- and 5-minute APGAR scores. The gestational age of the asphyxia group was mostly preterm. In addition, the mean level of CTnI was elevated in the asphyxia group, which is in accordance with a previous study.[9] Higher serum CTnI concentrations suggest that there is evidence of subclinical myocardial injury in perinatal asphyxia.

M-Mode echocardiographic parameters, encompassing left atrial dimension, MACS, LVEDD, LVESD, EF, FS, LV septum thickness, and LV posterior wall thickness, also showed significance in the asphyxia group. In the asphyxia group, EF was reduced, especially in the first 12 and 24 hours after birth. At 48 hours after birth, EF seemed to have slightly improved because of resuscitation and other clinical management techniques. TAPSE was reduced in the case group, which was indicative of RV dysfunction. In addition, 2D parameters such as LV posterior wall thickness, RV diastolic

dimension, and RV free wall thickness did not exhibit much difference between the 2 groups. Sobeih et al[11] (2019) reported that the M-Mode parameters of LVEDD and LVESD were higher in their asphyxia group than in their control group. Among TDI LV and RV parameters, only LV ejection time and RV ejection period/ejection time showed significance at 24 hours after birth ($P \leq 0.001$ and $P \leq 0.002$). Additionally, RV early diastolic wave showed significance at 24 and 48 hours after birth ($P = 0.001$ and $P < 0.01$, respectively). This result is in line with a previous study conducted by Matter et al.[12] The Tei index was higher in the asphyxia group, indicating biventricular dysfunction. Moreover, the Tei index was a more sensitive marker than the other markers of myocardial dysfunction in the asphyxia group. The Tei index has been reported to be the most useful tool in the assessment of global ventricular function independent of geometric assumptions, changes in preload or afterload, heart rate, and blood pressure.[13] A similar study conducted by Khattab[14] (2015) found that the Tei index was higher in neonates with perinatal asphyxia. A combination of the Doppler imaging of the mitral annulus and mitral inflow velocity curves provides a better estimation of LV filling pressures. As an individual parameter, the E/e' ratio showed a significant difference between the 2 groups at 48 hours after birth ($P < 0.001$). Moreover, we observed elevated left atrial pressure in the asphyxia group. Also, LV E/e' was higher among the asphyxia group on the second day of birth, depicting increased LV end-diastolic pressure among distressed neonates. The myocardial function assessment by TDI showed a significant reduction in the septal tissue annular velocity in the asphyxia group, which remained unaltered when assessed at 48 hours.

A larger cohort study may help predict maternal and fetal factors that would affect the neonatal outcome in asphyxia.

CONCLUSIONS

The asphyxia group in the current investigation showed a reduction in biventricular performance in comparison with healthy neonates. However, serial assessments revealed improvements in hemodynamics at 48 hours in response to standard medical therapy. Myocardial function assessments by using cardiac enzyme markers (CTnI) and different echocardiographic techniques appear to be more sensitive in the detection of ischemic cardiac injury in asphyxiated newborns.

Conflicts of Interest

None declared.

Acknowledgments

The first author prepared the draft of the manuscript, which was then reviewed and edited by the coauthors. All the authors actively contributed to the manuscript and approved the final version.

REFERENCES

1. Aslam HM, Saleem S, Afzal R, Iqbal U, Saleem SM, Shaikh MWA, et al. Risk factors of birth asphyxia. Italian journal of pediatrics. 2014;40(1):94.

2. Singh V, Vohra R, Bansal M. Cardiovascular involvement in birth asphyxia. Journal of Clinical Neonatology. 2018;7(1):20-.

3. Aslam HM, Saleem S, Afzal R, Iqbal U, Saleem SM, Shaikh MWA, et al. "Risk factors of birth asphyxia". Italian Journal of Pediatrics. 2014;40(1):94.

4. Lv H, Wang Q, Wu S, Yang L, Ren P, Yang Y, et al. Neonatal hypoxic ischemic

encephalopathy-related biomarkers in serum and cerebrospinal fluid. Clinica chimica acta. 2015;450:282-97.

5. Simovic AM, Prijic SM, Knezevic JB, Igrutinovic ZR, Vujic AJ, Kosutic JL. Predictive value of biochemical, echocardiographic and electrocardiographic markers in non-surviving and surviving asphyxiated full-term newborns. Turkish Journal of Pediatrics. 2014;56(3).

6. Gouda MH, Hammad BS, Amen MA. Short-term prognostic value of serum cardiac troponin I levels in neonates with perinatal asphyxia. Alexandria Journal of Pediatrics. 2017;30(3):108.

7. Koestenberger M, Friedberg MK, Nestaas E, Michel-Behnke I, Hansmann G. Transthoracic echocardiography in the evaluation of pediatric pulmonary hypertension and ventricular dysfunction. Pulmonary circulation. 2016;6(1):15-29.

8. Kanik E, Arun Ozer E, Rahmi Bakiler A, Aydinlioglu H, Dorak C, Dogrusoz B, et al. Assessment of myocardial dysfunction in neonates with hypoxic-ischemic encephalopathy: is it a significant predictor of mortality? The Journal of Maternal-Fetal & Neonatal Medicine. 2009;22(3):239-42.

9. Bhasin H, Kohli C. Myocardial dysfunction as a predictor of the severity and mortality of hypoxic ischaemic encephalopathy in severe perinatal asphyxia: a case–control study. Paediatrics and international child health. 2019;39(4):259-64.

10. Jehlicka P, Huml M, Rajdl D, Mocková A, Matas M, Dort J, et al. How to interpret elevated plasmatic level of high-sensitive troponin T in newborns and infants? Physiological research. 2018;67(2):191-5.

11. Sobeih AA, El-Baz MS, El-Shemy DM, Abu El-Hamed WA. Tissue Doppler imaging versus conventional echocardiography in assessment of cardiac diastolic function in full term neonates with perinatal asphyxia. The Journal of Maternal-Fetal & Neonatal Medicine. 2020:1-6.

12. Matter M, Abdel-Hady H, Attia G, Hafez M, Seliem W, Al-Arman M. Myocardial performance in asphyxiated full-term infants assessed by Doppler tissue imaging. Pediatr Cardiol. 2010;31(5):634-42.

13. Goroshi M, Chand D. Myocardial Performance Index (Tei Index): A simple tool to identify cardiac dysfunction in patients with diabetes mellitus. Indian heart journal. 2016;68(1):83-7.

14. Khattab AAA. Tei index in neonatal respiratory distress and perinatal asphyxia. The Egyptian Heart Journal. 2015;67(3):243-8.